PUBLIC HEALTH REPORTS

VOL. 49 JULY 20, 1934 NO. 29

PULMONARY INFECTION IN PNEUMOCONIOSIS

I. A BACTERIOLOGIC AND EXPERIMENTAL STUDY[1]

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INTRODUCTION

The constant physical irritation of the respiratory tract by inhaled dust particles may weaken the resistance of the mucous membranes and render them susceptible to the invasion of infectious microorganisms. In addition, the toxic influence of some dusts upon the tissues seems to be an important contributing factor to the development of respiratory infection.

Tuberculosis is the principal complication of silicosis, but pneumonia, lung abscess, bronchiectasis, and influenza are common in some pneumoconioses.

The ratio of the death rate in metal miners to that of the general population of California, Colorado, and Missouri, as calculated from the figures compiled by Sayers (1) for the years 1917 to 1920, was 5 to 1 for tuberculosis and 6 to 1 for pneumonia. Newer figures, according to Sayers and his coworkers (2), show that, in 1928, of the 7,722 metal miners examined in the Tri-State District of Oklahoma, Kansas, and Missouri, 1,914 had silicosis, and of that number 267, or 13.9 percent, had silicosis plus tuberculosis. In 1929, 8,853 men were examined in the same district (3). One thousand seven hundred and seventy-seven of these had silicosis, and of that number 220, or 12.4 percent, had silicosis plus tuberculosis. These figures are probably more representative than the more recent ones, because they are not influenced by the great shift of population which has occurred during the years of the economic depression.

It has been reported by some investigators (Gye and Kettle (4), Kettle (5), Collis (6)) that the products of cellular destruction make a most favorable medium for the proliferation of the tubercle bacillus. Kettle (7) has further shown, experimentally, that interstitial inoculation of different forms of silica produced characteristic necrotic

[1] From the Office of Industrial Hygiene and Sanitation. Special acknowledgment is made to the Metropolitan Life Insurance Co., which defrayed part of the expenses incurred in this study, and to the Bureau of Mines with whose cooperation this work was begun, and to Mr. W. P. Yant, Supervising Engineer, Bureau of Mines Experiment Station, Pittsburgh, Pa., and Surgeon F. V. Meriwether, United States Public Health Service, for their interest and assistance in the work.

lesions, and that the tubercle bacillus proliferated readily in such lesions in susceptible animals. Later he demonstrated that active growth of the bacilli did not depend merely on the necrosis of the tissue, but was actually due to the presence of the silica. He also showed, in experiments *in vitro*, that when silica is added to the culture medium, abundant growth is obtained in much shorter time than in its absence.

Gardner (8, 9) suggests that it is quite likely that inhaled dusts of certain types may of themselves light up a quiescent focus of infection, either through direct action on the bacilli or through altered cell metabolism with resulting formation of substances favorable to the growth of organisms.

The foregoing notes indicate that the relation of tuberculous infection to pulmonary dust disease has been studied to a considerable extent, but comparatively little investigative work has been attempted in connection with other infections, such as pneumonia, lung abscess, bronchiectasis, and influenza, conditions which are very often found in metal miners.

This study covers phases of the various infectious processes occurring in the pneumoconiotic lung, with the view to obtaining a better understanding of the predisposition to, and the mechanism of, infection.

FUSO-SPIROCHAETAL INFECTION OF THE LUNG

Pulmonary abscess and pulmonary gangrene, putrid bronchitis, and bronchiectases are of frequent occurrence among the miners in hard rock, particularly in those who have developed silicosis.

While making a survey of tuberculous infection among the miners of the Tri-State District, Cummings (10) called attention to the fact that the infection occurring in some of the men with silicosis was probably due to the fuso-spirochaetal group of organisms in symbiosis.

While it has been definitely established that the conditions may be caused by a variety of aerobic pathogenic organisms, such as staphylococci, streptococci, Friedländer bacillus, colon bacillus, certain types of fungi, etc., the anerobic microbes of the mouth have been found in preponderance in such a high percentage of the cases (Kline (11) and Smith (12)) that the possibility of their etiologic participation in these conditions must be seriously considered.

Until a few years ago little attention was paid to the frequent presence of spiral organisms and fusiform bacilli in pathologic material, except in non-diphtheritic ulcerations of the throat, e.g., Vincent's angina. Their responsibility for other, more serious infections of the respiratory system, however, was suspected.

Leyden and Jaffe (13) in 1867 had observed spiral organisms in the sputum and the pulmonary tissues of patients with putrid bronchitis

and pulmonary abscess and intimated their possible responsibility for the infection. Rona (14), in 1905, was probably the first European author who described in detail the presence of fuso-spirochaetal organisms in pulmonary gangrene. His work was followed in rapid succession by Feldmann (15), Küster (16), and Mühlens (17), in Europe, and by Johnson (18), Rothwell (19), and others in the United States. However, it was not until after the World War that the great importance of fuso-spirochaetal infections of the respiratory tract was thoroughly recognized. Since then several thousand cases of pulmonary abscess and gangrene of fuso-spirochaetal origin have been reported in the American literature and more are continually being added.

Accurate figures as to the incidence of putrid bronchitis and chronic bronchiectasis due to fuso-spirochaetal organisms are not available, but these conditions probably occur frequently. Smith (20) has demonstrated fusiform bacilli and spiral organisms in 80 percent of the cases of bronchiectasis studied by him over a period of 6 years at the New York State Hospital for Incipient Tuberculosis.

The mode of invasion of infectious organisms into the lungs has been described by Smith (21) as follows:

1. The organisms may be introduced directly through the chest wall, following stab wounds or gun-shot wounds in the thorax.
2. The infection may reach the lungs through the lymphatics.
3. The organisms may reach the lungs through the blood stream by means of an embolus from some other focus of infection in the body.
4. The lungs may be directly infected by aspiration from the upper respiratory tract.

MacCallum (22) states that infected emboli may reach the lungs and that such foci, at first hemorrhagic, will soon develop a gray, solid or rapidly liquefying center, surrounded by a zone of hemorrhagic, pneumonic consolidation, outside of which the lung is edematous. Such abscesses may reach a considerable size before coming in contact with a bronchus. This statement has been confirmed experimentally by Cutler (23, 24) and his collaborators.

The causative organisms may reach the lungs directly by aspiration from the upper respiratory tract. That this is readily accomplished has been demonstrated by a number of investigators in the United States and Europe (Hoelscher (25), Mullen and Ryder (26), Corper (27), Lemon (28)). Aspiration of infectious material from the throat is especially facilitated by anesthesia, but may also occur during sleep induced by narcotics and even during normal sleep (Iglauer (29) 1926, (30) 1928, May, Thoburn, and Rosenberger (31), Hamilton (32)).

Smith (33) believes that, because of the ease with which material from the mouth is aspirated into the lungs, and in view of the presence

of fuso-spirochaetal organisms in such a large percentage of lung abscesses, most of them, whether operative or nonoperative, are caused by aspiration of infected material from the mouth.

Lung gangrene is essentially a further development of lung abscess in which the causative bacteria are associated with the ordinary bacteria of putrefaction, which are, according to recent studies, a combination of fusiform bacilli and spirochaetal organisms (MacCallum (34)).

Bronchiectasis may be due to several causes. The bronchi may be partially or completely occluded in any part of their course by foreign bodies drawn into the larynx accidentally. Tumors, caseous lymph nodes, and aneurysms may also close the bronchus by pressure or by gradually growing into the lumen of the tube. Other forms of bronchiectasis may be acquired later in life and are in all cases associated with infection and inflammation of the bronchi. This in itself would not be sufficient cause for the widening of the bronchus until there is added a mechanical distention. The bronchus may be widened during acute inflammatory disease, but larger dilatations are produced more slowly by chronic processes and are often accompanied by profound changes of the surrounding tissues. McNeil and his collaborators (35) state that a bronchiectatic cavity is not a dilated bronchus, but an excavation in the lung substance starting in a bronchus.

The generally accepted theory for the explanation of the distention of the bronchi is that infection and inflammation weaken the bronchial wall and destroy its elasticity. Pleural adhesions generally accompany chronic bronchiectasis, and it has been suggested that in indurated and adherent lungs the contraction of the scar tissue between the bronchi pulls upon them from all sides and thus causes dilatation (MacCallum (36)).

In this report only those cases of lung abscess and bronchiectasis among hard-rock miners are considered which were caused by the anaerobic group of fusiform bacilli and spiral organisms probably aspirated from the oral cavity.

The average miner pays little attention to oral hygiene, with the result that a great many of them develop gingivitis and pyorrhea, for which the oral anaerobes are generally held responsible.

It has been stated before that bacteria may be directly aspirated into the deeper respiratory passages, where they are capable of setting up infectious processes if the microphages and the defensive mechanism are unable to cope with them.

Silica facilitates the growth of some organisms. The macrophage does not destroy ingested bacteria, because it is devoid of the endolysins which exert bactericidal action in other phagocytic leucocytes. Evidence also exists that living bacteria are protected through phagocytosis against the bactericidal substances in the blood and tissues

and are capable of spreading infection upon liberation from the carrier cell (37).

In consideration of these facts the following mechanism of infection would appear feasible:

The individual inhales large quantities of dust during the working hours which may become contaminated with the infectious material from the teeth and gums. It seems to occur more frequently among mouth breathers (Sayers et al. (2)).

The finer contaminated dust particles which have passed beyond the ciliated epithelium of the defensive system of the respiratory passages reach the alveoli, where they cause much catarrhal irritation and consequently produce favorable conditions for the proliferation of the organisms which they are carrying.

Some contaminated dust particles are engulfed by the so-called "dust cells" or macrophages and are removed from the alveoli to the lymph nodes at the end of the lymphatic channels along the alveolar ducts. Some are here deposited, while others proceed to the lymph nodes at the bifurcation of the bronchioles, the bronchi, and blood vessels, until they finally reach the various pulmonary lymph nodes. At these various points the heavily laden macrophages are deposited and may undergo degeneration. Fuso-spirochaetal organisms, which may have contaminated the dust particles, are thus set free and find a favorable environment in the degenerative tissue products. If represented in the proper symbiotic combination, they will be capable of setting up a fuso-spirochaetal infection.

In contrast to the characteristics of the bacillus of tuberculosis, which prefers to cause caseation around the fibrous patches, fusiform bacilli seem to invade these fibrous areas and cause necrosis and cavitations.

Finley and Graham (38) explain the development of bronchial dilatations in the fibrotic lung as follows: "To our mind the only feasible explanation of the condition is the fibrosis of the lung, as first suggested by Corrigan, with the added influence of pleural adhesions, as first pointed out by Hamilton, since without doubt by providing a fixed point for the contracting fibrous tissue these adhesions increase the traction force of the intrapulmonary fibrosis." This theory might well be applied to the majority of the cases of bronchiectasis in the silicotic miner.

Prolonged inhalation of silica dust, contaminated with fuso-spirochaetal material from the mouth, may cause continuous bronchial irritation and thus pave the way for bacterial attack upon the bronchial membranes, possibly resulting in putrid bronchitis and eventually bronchiectasis.

BACTERIOLOGY OF FUSO-SPIROCHAETAL LUNG DISEASE

The bacteriologic study of the anaerobic group of organisms of the respiratory tract was undertaken in the following manner:

Sputum samples were obtained from three cases of pulmonary abscess and from three cases of bronchiectasis with chronic putrid bronchitis in miners of the Tri-State Zinc and Lead Mining District in northeastern Oklahoma. The diagnosis had been established by clinical, roentgenologic, and bacteriologic examination at the United States Bureau of Mines cooperative clinic in Picher, Okla. Tuberculosis was ruled out. All cases were complicated with silicosis of the first and second stage.

The sputa were collected in sterile Petri dishes by one of the writers at the patients' homes, and great care was taken to avoid contamination with organisms from about the teeth and gums by instructing the patient to brush his teeth carefully and rinse his mouth and throat with copious amounts of sterile water. The sputum was then washed with several changes of sterile saline and immediately emulsified. A darkfield examination was made in every case in order to ascertain whether the material contained the representative groups of organisms, viz., spirochaetes, fusiform bacilli, cocci, and vibriones. One cubic centimeter of the sputum-saline emulsion was then immediately injected into the groin of guinea pigs. The tissues were not traumatized before inoculation. Sterile saline was used as a diluent in preference to serum water in order not to facilitate growth of the organisms in the presence of a foreign nutrient substance.

In addition, for comparative purposes, sputum containing fuso-spirochaetal organisms was obtained from a case of pneumoconiosis (silicosis and anthracosis) complicated with pulmonary moniliasis from the service of Dr. J. H. Barach at the Presbyterian Hospital in Pittsburgh, Pa., and from 7 cases of uncomplicated bronchiectasis of 2 to 45 years' standing. The latter cases had never been exposed to harmful dusts and were available through the courtesy of Dr. C. H. Marcey, of the Tuberculosis League Hospital of Pittsburgh.

The sputa obtained in Pennsylvania were collected and treated in the same manner as were those from the Oklahoma miners, and 1 cubic centimeter was injected into the groin of guinea pigs. The remainder of the specimens were placed under anaerobic conditions and reserved for further examination in the laboratory.

Most animals developed large necrotic abscesses at the site of injection within 4 or 5 days which, upon puncture, yielded a greenish-gray, extremely foul-smelling pus and contained, in addition to vast numbers of other bacteria, several types of spirochaetes, fusiform bacilli, cocci, spirilla, and vibriones which morphologically resembled those of the original material. Some of these abscesses contained considerable malodorous gas which would push the piston from the

barrel of the aspiration syringe. In these cases a small amount, usually 0.25 cubic centimeter, of pus was injected into the groin of a fresh guinea pig, and this procedure continued until the gas-producing organisms were eliminated. Three to four passages would usually accomplish this, and the resulting purulent material was used for the cultural procedures.

The anaerobic organisms which were constantly recovered from the pathological material described above were the following:

Treponema microdentium (Noguchi), *Treponema mucosum* (Noguchi), *Treponema macrodentium* (Noguchi), *Treponema buccale* (Lühe-Dobell), *Treponema vincenti* (Blanchard), *Bacillus fusiformis* (Vincent), *Spirillum sputigenicum* (Miller), *Vibrio viridans* (Miller), and anaerobic streptococci.

The identification of the anaerobic, fuso-spirochaetal organisms by their morphologic characteristics is not difficult and may be readily accomplished with the aid of a good darkfield attachment to the microscope, or by appropriate staining; but their isolation in pure culture presents many difficulties. The methods used are discussed under the heading "Technical Considerations."

Excellent descriptions and illustrations of the morphologic and cultural characteristics of the organisms under consideration may be found in the various special treatises and textbooks on bacteriology. Hence, the present discussion will be confined to a brief résumé of the observations and measurements made in this investigation, together with a few notes on the immunologic behavior of the organisms.

Treponema microdentium

The organism was described by Noguchi (39) in 1912.

Morphology.—Delicate spiral filament with regular, shallow convolutions. Length 4 to 8 μ, width 0.25 μ and less. In pathologic material and in young cultures the short forms predominate; in older cultures longer forms are found in abundance, often measuring up to 15 μ. The organism tapers sharply toward the extremities, and occasionally delicate caudal filaments may be observed on one or both ends. The latter are, however, not independently motile, but rather seem to be propelled by the organism itself and cannot therefore be looked upon as true flagella. Granular particles have been repeatedly observed. Motility is active and rotating. The body is flexible. Division is transverse, but Noguchi has also seen longitudinal fission.

Culture.—The organism is strictly anaerobic. Growth in ascitic agar and heart infusion agar stabs begins within 48 hours at 37° C. Colonies are rather dense and whitish, with distinct outer boundaries and grow away from the stab canal into the clear medium. No gas is formed. A fetid odor gradually develops in pure cultures.

Pathogenicity.—The organism is nonpathogenic for laboratory animals when washed cultures are injected; but causes slight, transient inflammations when injected in ascitic agar emulsion.

Immunology.—The organism agglutinates in homologous antiserum and cross-agglutinates in T. mucosum antiserum to a slight but distinct extent.

Treponema mucosum

Described by Noguchi (39) in 1912.

Morphology.—The organism is slightly longer and thicker than *T. microdentium*. Length 8 to 12 μ, width 0.25 to 0.3 μ. The coils are very regular and quite deep. The ends are sharply pointed and often possess finely curved terminal projections of varying length. True flagella have not been observed. The body is flexible. Motility is quite active and rotating. Division is transverse. Noguchi, however, claims to have seen longitudinal fission. Fine granules are occasionally present.

Culture.—The organism grows well in ascitic agar and heart infusion agar stabs, even without the addition of sterile tissue, but is strictly anaerobic. Growth begins within 48 hours at 37° C. as a whitish cloud in the lower two-thirds of the tube. Outer boundaries of the colonies are indistinct. Gas is not formed. The organism produces a strong fetid odor in pure cultures which is much more offensive than that produced by *T. microdentium*. In older cultures, in ascitic agar, *T. mucosum* produces varying amounts of tenacious mucus; hence its name. It is quite resistant and will remain viable in cultures for many weeks. Upon subculturing it gradually loses its mucin-producing qualities, but its odor-producing properties persist.

Pathogenicity.—The organism is nonpathogenic for laboratory animals when washed cultures are injected, but produces acute, transitory inflammation if injected in ascitic agar emulsion.

Immunology.—The organism agglutinates in homologous antiserum and cross-agglutinates moderately in *T. microdentium* antiserum.

Treponema macrodentium

Described by Noguchi (40) in 1912.

Morphology.—The organism is much larger than either *T. microdentium* or *T. mucosum*. Length 4 to 15 μ, width 0.3 to 0.35 μ. In young cultures, short plump individuals are often seen which have from 3 to 6 convolutions and measure from 0.6 to 1.0 μ in width; these forms appear often doubly contoured in the dark field, and resemble *Treponema buccale*. They do not belong to a separate species, because they disappear in older cultures and give rise to normal forms. The extremities of mature organisms taper more gradually than do those of *T. microdentium* and *T. mucosum*. The curves are very regular

and are more shallow than those of the other mouth treponemata. The body is flexible. Motility is agile and rotating. Terminal filaments and flagella have not been observed. Division is transverse. Granular phase is present.

Culture.—The organism is a strict anaerobe and by far the most difficult to isolate. Growth in ascitic agar and heart infusion agar stabs begins in from 48 to 72 hours at 37° C. and appears as a faint, almost transparent turbidity, diffusing into the clear medium. There is no line of demarcation at the periphery of the colonies. No odor or gas is produced in pure cultures. Odor is always evidence of contamination with other mouth spirochaetes.

Pathogenicity.—The organism is nonpathogenic for laboratory animals.

Immunology.—The organism agglutinates in homologous anti-serum but does not cross-agglutinate in *T. microdentium* or *T. mucosum* anti-sera.

Complete plasmolysis occurs with all three types of treponemata in 10-percent to 1-percent solutions of either sodium taurochloate or saponin.

Two very small types of mouth treponemata have been reported by Hoffmann (41), which he named *Treponema orthodontium* and *T. skoliodontium*. These organisms are smaller than the smallest types of treponemata. They have never been encountered in the material under examination in this work or else have not been recognized from morphologic and cultural characteristics.

The same author (Hoffmann (42)) has also observed organisms of the genus *Leptospira* (*Leptospira trimerodonta*), but these seem to be extremely rare in the United States. Smith (43), upon examination of over 100 mouths during a period of 5 years, has encountered them once, and Ermatinger (44) reported *Leptospira* in one case of pulmonary abscess. One of the writers has examined over 2,000 mouths of men, women, and children of the Aryan, American Indian, and Ethiopian races and has never found *Leptospira*.

Treponema buccale

Treponema buccale (Lühe-Dobell) was orginally described by Cohn (45) in 1875.

Morphology.—The organism is a spiral filament of rather large dimensions. Length 5 to 20 μ, width 0.6 to 0.8 μ. The thin specimens (0.3 μ) described in Ford's textbook (46) was probably identical with *Treponema vincenti*. When viewed with the darkfield apparatus, *T. buccale* presents doubly contoured outlines. The ends are sharply pointed and devoid of flagellar extensions. The curves are wide and irregular. Motility is generally sluggish and undulating. Noguchi suggests that the organism may be a *Spironema* rather than a *Treponema*.

Culture.—The organism is strictly anaerobic and very difficult to culture. It multiplies in deep agar stabs, but does not leave the stab canal or penetrate the clear agar like the other treponemata. Smith (47) succeeded in growing *T. buccale* upon the surface of blood agar plates on which the colonies appear as a flat growth, 1 to 2 millimeters in diameter, under which the blood has assumed a brownish discoloration. The colonies are best seen by reflected light. It was possible to duplicate Smith's results on this medium and also to grow the colonies upon ascitic-hormone agar and "cooked blood" agar plates. Upon the latter, the colonies appear more distinct than on blood agar and develop a flat, grayish, irregular, colony.

Pathogenicity.—The organism is non-pathogenic for laboratory animals.

Immunology.—Immunologic experiments have not been made as yet, because of the meager yield of culture material.

Treponema vincenti (Blanchard)

First described by Plaut (48) and Vincent (49).

The biologic position of this organism has not been definitely established and has given rise to much controversy and speculation. Some investigators regard it as an independent species; others look upon it as a transitional form in the life cycle of a large fusiform bacillus. Tunicliff (50) describes such a life cycle of *Bacillus fusiformis* in which the spirochaetes developed from an apparently pure culture. One of the writers made similar observations in cultures of certain *fusiform* bacilli, in which the latter gradually disappeared and *motile* spirals developed. Subcultures however, failed to show growth. It is possible that the disintregration products of the bacilli are necessary for the propagation of the spirochaetes. Experiments are now under way to test this theory and to study the problem in cultures which have been started from a single organism.

Morphology.—The organism varies in size, the majority being from 6 μ to 10 μ in length and from 0.3 μ to 0.5 μ in width. The thicker and less motile organisms described by some authors were probably *T. buccale.* The coils are shallow and irregular, and motility is quite active. Motion is serpentine rather than rotating. Granules have been frequently observed.

Culture.—The organism has not been obtained in pure culture, and the animal experiments described in another section of this paper have been carried out with culture of fusiform bacilli in which spirochaetes had developed.

Pathogenicity.—Such mixed cultures were nonpathogenic for laboratory animals.

Immunology.—Immunologic experiments have not been attempted.

Bacillus fusiformis

Several types of fusiform bacilli have been recovered from the respiratory tract, two of which were predominantly and consistently found in the pathologic material under examination. These two types corresponded to the serologic type I, subtype 2, and type II of Varney (51).

TYPE I, SUBTYPE 2 (Varney)

Morphology.—Length 6 to 12 μ, width 0.5 to 0.7 μ. The ends are sharply pointed. Single organisms are straight, but when occurring in pairs (tandem form) they present a slightly curved appearance. In this case the apposed ends are blunt. Occasionally short chains of three to four individuals are formed. The organism is often gathered in bundles. Fine granules (from 2 to 6) may be frequently seen. It is nonmotile, but in fresh pathologic material it often exhibits a vibratory motility. The bacillus is devoid of flagella. Division is transverse. It behaves negatively toward Gram's stain

Culture.—The organism is an obligate anaerobe and very fastidious in regard to culture media. Blood or ascitic fluid is essential for satisfactory growth. Dextrose may be omitted. A little sodium citrate often facilitates growth. Colonies upon satisfactory media appear in from 48 to 72 hours at 37° C. They are white, round, and slightly viscid. Diameter about 1 millimeter. Pure cultures give off a faint, not unpleasant odor. No hemolysis is produced upon blood agar.

Pathogenicity.—The organism is nonpathogenic for laboratory animals.

Immunology.—Agglutination takes place in homologous antiserum. Slight, but distinct cross-agglutination occurs in antiserum type II of Varney.

TYPE II (Varney)

Morphology.—This bacillus is smaller than the one described above. It occurs singly and in tandem form. It does not form chains, but frequently gathers in bundles. The ends are sharply pointed. Length 3 to 5 μ, width 0.3 to 0.4 μ. The organism is nonmotile and nonflagellate. Granules have not been observed. It is Gram-negative.

Culture.—Colonies are rounded, raised, gelatinous, and adherent to the medium, on which they leave slight depressions. Diameter from 1 to 2 millimeters. Pure cultures give off a slight, not unpleasant odor. No hemolysis is formed on blood agar.

Pathogenicity.—The organism is nonpathogenic for laboratory animals.

Immunology.—It agglutinates in homologous anti-serum and cross-agglutinates in type I, sub-type 2 anti-serum.

Spirillum sputigenum

Described by Miller (52, 53) in 1892 and 1906.

Morphology.—The organism is large and comma or crescent shaped, with the latter form predominating. Length 2 to 6 μ, width 0.5 to 0.8 μ. It often combines in pairs to form the letter S. Chains, composed of more individuals, simulating spirochaetes, are frequently seen, but are readily identified by their mode of locomotion. In specially stained preparations from one to three flagella may be demonstrated, which are located laterally on the concave side. The normal number of flagella is apparently three; individuals with less evidently have lost them in the preparation of the specimen, as evidenced by the numerous unattached filaments usually found on the slide. Motility is active, vibratory, rotating, and whirling. The organism is Gram-negative.

Culture.—Growth is successful in ascitic-harmone agar stabs, in which it appears in from 48 to 72 hours at 37° C., under strictly anaerobic conditions. The colonies are hazy, with an opaque grayish or yellowish center, and are located just outside the stab canal, in the neighborhood of the tissue, in the depth of the medium. Addition of dextrose is unnecessary. Surface colonies on blood agar plates have as yet not been obtained. Odor is not produced.

Pathogenicity.—The organism is non-pathogenic for laboratory animals.

Immunology.—No serologic experiments have been attempted, because of the ease with which the organism may be recognized morphologically.

Vibrio viridans

Described by Miller (54) in 1890.

Several types of anaerobic vibriones have been reported in the literature. The one found consistently in the pathologic material available, resembled most closely that described by Miller (54) and by Smith (55).

Morphology.—The organism is short, plump, and polymorphic. The smaller cells are coccoid, while the larger ones are slightly invaginated on one side. They are provided with from 1 to 3 long, fine, terminal flagella. Here, as in the case of *Spirillum sputigenum*, the presence of numerous free flagella in the stained preparation suggests that the normal number of flagella may be 3. Frequently the flagella are entangled and in that form simulate spirochaetes or hypertrophied flagella. Motility is extremely active, vibratory, and whirling. Ford very realistically likens this motility to the swarming of midges. The organism is Gram-negative and stains readily with dilute carbol fuchsin and gentian violet.

Culture.—The organism is a "transitory" anaerobe. By this is meant that it loses its anaerobic characteristic upon subculturing. It grows upon blood agar and ascitic hormone agar, usually together with cocci, but may be quite easily separated from them. Colonies are round, moist, and raised; diameter 1 to 2 millimeters. The organism causes a greenish-gray discoloration upon blood agar. Odor is not produced by pure cultures.

Pathogenicity.—It causes local swelling in guinea-pigs when inoculated subcutaneously, but is not pyogenic.

Immunology.—A serologic classification of the vibrios of the mouth is being attempted by one of the writers and will be reported later.

Streptococci

In all cases of fuso-spirochaetal lung disease investigated in the course of this work, a short-chain streptococcus was encountered which at first would grow only under anaerobic conditions. Smith (55) describes an anaerobic *hemolytic* streptococcus which he isolated from his series, and Cohen (56) found an anaerobic anhemolytic streptococcus in the material from a number of pulmonary abscesses. The organism found by one of the writers corresponded to the latter.

Morphology.—The streptococcus occurs in short chains of from 4 to 8 cells, slightly longer in liquid media.

Culture.—The organism, at first strictly anaerobic, loses this characteristic after 2 or 3 transplantations. It grows readily upon blood agar plates made anaerobic by the pyrogallic acid-sodium hydroxide method or the pyrogallic acid–Rockwell (57) method. It grows frequently mixed with vibriones. The possibility of contamination with an aerobic streptococcus was ruled out by single-cell cultivation. It produces a grayish colony upon blood agar.

The organism ferments dextrose, saccharose, and maltose, but does not split inulin and is not plasmolyzed by bile.

Pathogenicity.—The organism causes transitory inflammatory reaction when inoculated subcutaneously into laboratory animals.

Immunology.—Immunologic experiments have not as yet been made.

Several other species of anaerobes from the upper respiratory system have been reported in the literature, but they do not enter into the symbiotic combination with which the experimental lesions, to be described in a later report, were produced, and for that reason they will be merely mentioned, together with their original references:

Bacillus gonidiaformans—Tunicliff and Jackson (58),

Bacterium melaninogenicum—Oliver and Wherry (59),

Lepthothrix buccalis—Robin (Trevison) (60),

Micrococcus gazogenes alkalescence anaerobicus—Lewkowicz (61),

Vibriothrix tonsillaris—Tunicliff (62).

TECHNICAL CONSIDERATIONS

It has been stated before that the morphologic identification of the fuso-spirochaetal organisms is not difficult, provided that a good darkfield attachment for the ordinary microscope is available. The India ink method may be used, but is not very dependable.

For the preparation of stained specimens, the Fontana silver method is very convenient, but the reagents are not stable; the organisms appear thicker than they really are, owing to the coating of reduced silver, and the preparations will fade rapidly.

The following staining method has given very satisfactory results:

Solution I:	*Cubic centimeters*
Ammonium (potassium) alum, saturated aqueous solution	20
Tannic acid, saturated alcohol solution	20
Basic Fuchsin, saturated alcohol solution	5

Mix, filter, and allow to stand over night.

Solution II: Loeffler's methylene blue.

Dilute solution I with 4 parts of distilled water. Cover the air-dried preparation with the diluted stain and steam gently for 1 minute. Wash with distilled water and counterstain with solution II for 1 minute. Blot with filter paper. Spirochaetes appear purplish, flagella blue. For photographic purposes omit counterstaining.

In culturing fuso-spirochaetal organisms the culture methods of the original investigators will, of course, yield the best results with the organism for which they were specifically devised. This will, however, necessitate the use of a number of special media of varying composition, proportion, and hydrogen-ion concentration. For routine fuso-spirochaetal work it is desirable to have available one basic medium which incorporates all of the factors essential for the growth and propagation of the whole group of organisms, and which can be adapted for the individual species by simple manipulation. After extensive and careful experimentation it was found that the following media answered that purpose and gave most satisfactory results:

Agar base.—This is essentially a modification of Huntoon's (63) "Hormone" agar made with veal heart infusion from which the growth-promoting factors have been extracted in the presence of a colloidal suspension of agar. It is prepared as follows:

Veal heart, freed from fat and blood vessels, finely ground	grams	500
Bacto peptone	do	10
Sodium chloride (c.p.)	do	5
Bacto agar, powdered or granular	do	18
Albumin from 1 egg.		
Distilled water (tap water should not be used)	cubic centimeters	1,000

The mixture is stirred well and placed in the refrigerator for 18 hours. It is then heated in a double boiler to 70° C., or until it turns brown. Normal sodium hydroxide is added until slightly alkaline to litmus. Steam in the Arnold sterilizer for 1 hour. Loosen the coagulum from the walls of the vessel and return to the sterilizer for another hour. Siphon off the hot clear liquid and adjust the reaction to pH 7.6. Distribute the medium in 100-cubic centimeter quantities into Erlenmeyer flasks. Final sterilization is carried out by the fractional method. The reaction of the finished medium will be about pH 7.4. Filtration must be avoided; clearing may be accomplished by centrifugation while the medium is still hot.

A liquid medium of similar composition may be obtained by reducing the amount of agar to 10 grams per liter.

Ascitic fluid.—Not all specimens of ascitic fluid are suitable for bacteriologic work, and cloudy or green fluids should be rejected. It is advantageous to pool 2 or 3 samples. The pH concentration should be determined; and if the fluid is too alkaline, the agar base should be correspondingly acidified to obtain a final pH of 7.4.

For stab cultures one part of ascitic fluid is added to two parts of heart infusion agar. Tall test tubes should be filled with this mixture to a depth of at least 10 centimeters. A piece of fresh sterile rabbit or guinea-pig kidney, about the size of 1 cubic centimeter, is placed in the bottom of the tube. It is important that the medium be mixed just before use.

Sterile rabbit's blood, citrated.—It appears that some of the fusiform bacilli grow better in the presence of a little citrate, which does not interfere with the growth of the other anaerobes. For blood agar plates 4 percent of the sterile citrated rabbit's blood is added to the melted agar base. The addition of dextrose may be omitted. For "cooked blood" agar plates, 4 percent of the sterile citrated rabbit's blood is added to the boiling hot agar base.

The pathologic material, sputum and pus, is collected as described elsewhere. The emulsion should be diluted with sterile saline to a slight opalescence and a droplet examined in the darkfield to ascertain that all fuso-spirochaetal organisms are represented.

Not less than 10 tubes should be inoculated by the stab method. A fine glass capillary pipette is preferred rather than the customary platinum wire, because with the latter the inoculum is often confined to the upper one-third of the agar column and very little reaches the depth of the medium near the tissue, where anaerobic conditions are most favorable.

Blood agar plates are seeded with the aid of a heavy gauge platinum wire. Bent glass rods are not practical. Special inoculating machines and inoculating brushes are unnecessary, but inoculation should be made with firm pressure, so as to produce shallow grooves upon the agar surface, yet without tearing the medium.

Anaerobic conditions may be created by any of the various methods in vogue and in any suitable container available. One of the writers in his work used the pyrogallic acid-sodium hydroxide method exclusively, with satisfactory results. For test-tube cultures Wright's method was employed, and for plate cultures a simple modification of that of Lentz (64) as follows: Glass plates, 12 by 36 centimeters in diameter, are cut and sterilized. Three rings of plasticine are made on each plate, corresponding to the diameter of the inner part of a Petri dish. Into this well are measured 10 cubic centimeters of 1 percent sodium hydroxide solution. Three sheets of filter paper, 9 centimeters in diameter, which have been previously saturated with a strong alcoholic solution of pyrogallic acid and dried, are placed in the hydroxide solution, and the inoculated, inverted Petri dish is immediately pressed upon the plasticine ring, which will effect a perfect seal, while the pyrogallic acid paper slowly absorbs the alkali. The excess of plasticine on the outer edge of the dish is tightly smoothed against the base plate with a spatula. This simple method has several advantages over the jar methods, viz:

1. The inoculated plates are quickly warmed to incubator temperature, promoting immediate bacterial growth.

2. Sufficient but no excessive moisture is derived from the dilute sodium hydroxide.

3. The plates may be examined daily without the necessity of opening and resealing a cumbersome jar. Pyrogallic acid papers may be kept on hand and the plates may be resealed without much inconvenience.

4. The method is economical, because it does not require expensive apparatus and large quantities of absorption material.

5. The plates may be stacked and therefore take up much less room in the small incubator than large anaerobic jars.

After well-defined colonies of spirochaetes have developed in the depth of the cultures, along the stab canal, the following convenient method for removing them has given good results: The paraffin seal is carefully removed from the top of the agar. A long, wide, sterile, glass capillary is introduced alongside the stab canal and passed through a colony. This will punch out a thin agar column containing part of the colony. Care should be taken not to come too close to the stab. The capillary is then broken at the location of the colony and the growth is removed and emulsified in a little sterile saline. A dark-field examination will show whether a pure culture has been obtained. From the resulting spirochaetal emulsion, subcultures may be seeded in fresh media. This simple procedure eliminates the danger of accidents to the worker which so often accompany the breaking of the heavy culture tube, and also prevents contamination with organisms

from the stab canal. Another advantage is that the original culture is not destroyed, but may be resealed and returned to the incubator for further growth development. With a little practice, much time will be saved by eliminating subculture periods in purifying mixed colonies.

All pure cultures are conveniently maintained in deep agar stabs instead of upon the surface of agar slants and plates.

SUMMARY

In general, the silicotic lung is more susceptible to bacterial infection than the average lung. This is probably due to the irritation of the respiratory tissues by the inhaled dust particles which weakens the mucous membranes and renders them susceptible to infection. The toxic influence of certain inorganic dusts upon the tissues may be a contributing factor.

The relation of tuberculosis to pneumoconiosis has been studied to a considerable extent, but comparatively little work has been done in connection with other infectious processes of the lung, e.g., pneumonia, lung abscess, bronchiectasis, and influenza. An investigation was made of these conditions, both bacteriologically and experimentally, with the view of obtaining a better understanding of the predisposition to, and the mechanism of, infection of the lung in certain dusty trades.

Bronchiectasis, lung abscess, and gangrene occur frequently in hard-rock miners. It has been definitely established that aerobic, pathogenic bacteria, and certain fungi are responsible for these conditions, but the high percentage of cases in which the anaerobic microbes of the mouth and throat have been reported would suggest that they at least participate in the etiology of the diseases.

The responsibility of fuso-spirochaetal organisms for severe infections of the respiratory tract had been suspected as early as 1867. In the past few years more than 2,000 cases of fuso-spirochaetal lung abscess have been reported in the United States. Accurate figures of bronchiectasis of the same origin are not available.

The mode of infection of the lungs in individuals not engaged in dusty trades is briefly discussed and compared with the possible mechanism of infection in those having pneumoconiosis.

The bacteriology of fuso-spirochaetal lung disease is described in detail and a practical technique for the study of the anaerobic flora of the upper respiratory tract is appended.

REFERENCES

(1) Sayers, R. R.: Silicosis among miners. U.S. Bureau of Mines Technical Paper No. 372.

(2) Sayers, R. R., Meriwether, F. V., Lanza, A. J., and Adams, W. W.: Silicosis and tuberculosis among miners of the Tri-State District of Oklahoma, Kansas, and Missouri. I. For the year ended June 30, 1928. U.S. Bureau of Mines Technical Paper No. 545.

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(3) Meriwether, F. V., Sayers, R. R., and Lanza, A. J.: Silicosis and tuberculosis among miners of the Tri-State District of Oklahoma, Kansas, and Missouri. II. For the year ended June 30, 1929. U.S. Bureau of Mines Technical Paper No. 552.
(4) Gye, W. E., and Kettle, E. H.: The pathology of miner's phthisis. Lancet, 2:855 (1922).
(5) Kettle, E. H.: Experimental silicosis. Jour. Ind. Hyg., 8:491 (1926).
(6) Collis, E. L.: The statistical characteristics of dust phthisis (pulmonary silicosis). Jour. Ind. Hyg., 8:457 (1926).
(7) Kettle, E. H.: The relation of dust to infection. Proceedings of the Royal Society of Medicine (section of Pathology), 24:1 (1930).
(8) Gardner, L. U.: Studies on experimental pneumonokoniosis. V. The reactivation of healing primary tubercles in the lung by the inhalation of quartz, granite, and carborundum dusts. Am. Revue Tuberculosis, 20:833 (1929).
(9) Gardner, L. U.: Will the inhalation of siliceous dust activate a partially healed focus of tuberculous infection? Pub. Health Rep., 45:282 (1930).
(10) Cummings, Donald E.: Personal communication.
(11) Kline, B. S., and Berger, S. S.: Pulmonary abscess and pulmonary gangrene. Arch. Surg., 18: pt. I, 481 (1929).
(12) Smith, D. T.: Oral spirochetes, p. 153. The Williams and Wilkins Co., Baltimore. 1932.
(13) Leyden, E., and Jaffé, M.: Ueber Putride (Foetide) Sputa nebst einigen Bemerkungen über Lungenbrand und Putride Bronchitis. Deutsch. Arch. für klin. Medizin, 2:488 (1867).
(14) Rona, S.: Zur Aetiologie und Pathogenese der Plaut-Vincentschen Angina. Arch. für Dermatol. und Syphil., 74:171 (1905).
(15) Feldmann, Ignàcz: Beiträge zu den durch Bac. fusiformis und Spirillum dentium hervorgerufenen Infektionen mit besonderer Berücksichtigung der Eiterungen. Wien. klin. Wochenschr., 19:695 (1906).
(16) Küster, E.: Demonstration von Spirochaeten beim Gangraena pulmonum. Verh. Deutscher Naturforscher und Aerzte, Dresden, vol. 74 (1907).
(17) Mühlens, P.: Vergleichende Spirochätenstudien. Zeitschr. f. Hyg., 52: 405 (1907).
(18) Johnson, W. B.: A case of spirillosis of the lung. Memphis Med. Month., 29:183 (1909).
(19) Rothwell, J. H.: Bronchial Vincent's angina. Jour. Amer. Med. Assoc., 54:1867 (1910).
(20) Op. cit. Reference 12, p. 153.
(21) Op. cit. Reference 12, p. 116.
(22) MacCallum, W. G.: Textbook of pathology, 4th edition, p. 254. W. B. Saunders Co., Philadelphia.
(23) Cutler, Elliott, C.: Studies in the experimental production of abscess of the lung. Jour. Coll. of Surgeons of Australasia. 2:201 (1929–30).
(24) Cutler, Elliott, C.: The etiology of postoperative abscess of the lung. Ohio State Med. Jour., 24:109 (1928).
(25) Hoelscher, R.: Experimentelle Untersuchungen über die Entstehung der Erkrankungen der Luftwege nach Aethernarkose. Arch. f. klin. Chir. 57:175 (1897).
(26) Mullen, W. V., and Ryder, C. T.: Experimental lesions of the lung produced by the inhalation of fluids from the nose and throat. Am. Rev. Tuberc., 4:683 (1920).
(27) Corper, H. J.: Pulmonary aspiration of particulate matter, normally and during anesthesia. Jour. Amer. Med. Assoc. 78: pt. II, 1858 (1922).

(28) Lemon, W. S.: Aspiration. Experimental study. Arch. Surgery, 12: 187 (1926).
(29) Iglauer, S.: Injected iodized oil in roentgen-ray diagnosis of laryngeal, tracheal, and broncho-pulmonary conditions. Jour. Amer. Med. Assoc., 86: 1879 (1926).
(30) Iglauer, S.: Aspiration of blood into the larynx and trachea during tonsillectomy under local anesthesia. Ann. Otol., Rhinol., and Laryng., 37:23 (1928).
(31) May, R. V., Thoburn, T. W., and Rosenberger, H. C.: Aspiration during tonsillectomy. Jour. Amer. Med. Assoc., 93: 589 (1929).
(32) Hamilton, R. L.: Non-tuberculous broncho-pulmonary infection, from the internists point of view. Canadian Med. Assoc. Jour., 23: 30 (1930).
(33) Op. cit. Reference 12, p. 117.
(34) Op. cit. Reference 22, p. 248.
(35) McNeil, MacGregor, and Alexander, cit. by Erb, I. H.: Pathology of bronchiectasis. Arch. Pathol., 15: 357 (1933).
(36) Op. cit. Reference 22, p. 406.
(37) Wells, H. G.: Chemical pathology, p. 266. (Citing Schneider, Rous, and Jones). 3d. ed., W. B. Saunders Co., Philadelphia and London.
(38) Finlay, L., and Graham, S., cit. by Erb, I. H.: Pathology of bronchiectasis. Arch. Pathol., 15: 357 (1933).
(39) Noguchi, Hideyo: Cultural studies on mouth spirochaetae (Treponema microdentium and macrodentium). Jour. Exper. Med., 15: 81 (1912).
(40) Noguchi, Hideyo: Treponema mucosum (n.sp.). A mucin-producing spirochaete from Pyorrhea alveolaris, grown in pure culture. Jour. Exper. Med., 16: 194 (1912).
(41) Hoffmann, Erich: Ueber eine der Weilschen Spirochäte ähnliche Zahnspirochäte des Menschen. Deutsche med. Wochenschr., 46: 257 (1920).
(42) Hofmann, Edmund: Einige Bemerkungen über die Leptospira dentium-Hoffmann und andere Mundspirochäten. Centralbl. f. Bakteriologie, Orig., 86: 134 (1921).
(43) Op. cit. Reference 12, p. 7.
(44) Ermatinger, L. H.: Micro-organisms of lung abscess and bronchiectasis. Jour. Inf. Dis., 43: 391 (1928).
(45) Cohn, Ferdinand: Beiträge zur Biologie der Pflanzen, 3: 199 (1875).
(46) Ford, W. W.: A textbook of bacteriology. W. B. Saunders Co., Philadelphia.
(47) Op. cit. Reference 12, p. 11.
(48) Plaut, H. C.: Studien zur Bakteriellen Diagnostik der Diphtherie und der Anginen. Deutsche med. Wochenschr., 20: 920 (1894).
(49) Vincent, H.: Sur l'etiologie et sur les lésions anatomopathologiques de la pourriture d'hospital. Ann. de l'Institut Pasteur, 10: 488 (1896).
(50) Tunicliff, Ruth: The life cycle of Bacillus fusiformis. Jour. Inf. Dis., 33: 147 (1923).
(51) Varney, P. L.: The serological classification of fusiform bacillus. Jour. Bact., 13: 275 (1927).
(52) Miller, W. D.: Die Mikroorganismen der Mundhöhle. Leipzig, 1892.
(53) Miller, W. D.: Ueber eine scheinbar pathogene Wirkung der Spirochaete dentium. Deutsche med. Wochenschr., 32: I, 348 (1906).
(54) Miller, W. D.: The micro-organisms of the human mouth. S. S. White Dental Manuf. Co., Philadelphia, 1890.
(55) Smith, D. T.: Fuso-spirochaetal disease of the lungs produced with cultures from Vincent's angina. Jour. Inf. Dis., 46: 303 (1930).
(56) Cohen, J.: Bacteriology of abscess of lung and method for its study. Arch. Surg., 24: 171 (1932).

(57) Rockwell, cit. by Stitt, E. R.: Practical bacteriology, bloodwork, and Animal Parasitology, 8th ed., p. 83. P. Blakiston Son Co., Philadelphia.
(58) Tunicliff, R., and Jackson, L.: Bacillus gonidiaformans (n.sp.). Jour. Inf. Dis. 36: 430 (1925).
(59) Oliver, W. O., and Wherry, W. B.: Notes on some bacterial parasites of the human mucous membrane. Jour. Inf. Dis., 28: 341 (1921).
(60) Robin, Charles: Histoire naturelle des végétaux parasites qui croissent sur l'homme et sur les animaux vivants, p. 42. Paris, 1847; and
Vignal, W.: Recherches sur les micro-organismes de la bouche. Arch. de physiologie normale et pathologique, 18: 325 (1886).
(61) Lewkowicz, Xavier: Recherches sur la flore microbienne de la bouche des nourrissons. Arch. de Med. Expér. et d'Anat. Patholog., 13: 633 (1901).
(62) Tunicliff, R., and Jackson, L.: Vibriothris tonsillaris. Jour. Inf. Dis., 46: 12 (1930).
(63) Huntoon, F. M.: "Hormone" medium. A simple medium employable as a substitute for serum medium. Jour. Inf. Dis., 23: 169 (1918).
(64) Lentz, cit. by Kolle and Hetsch: Die experimentelle Bakteriologie und die Infektionskrankheiten, Band 2, Aufl. 5, p. 1333. Urban und Schwarzenberg, Berlin.

CALIFORNIA HEALTH DEPARTMENT REGULATION REGARDING CONVALESCENT SERUM

Following the death of one child, age 2, and the severe illness of his brother, age 7, in California in the latter part of June, resulting from the prophylactic use of human serum, the State department of public health issued a regulation restricting the manufacture and distribution of human convalescent serum to laboratories that are approved by the State department of health. The regulation reads as follows:

Whereas, it appears that there has been distributed by certain laboratories, improperly prepared convalescent and other serum, taken from human beings for use in the prevention and treatment of persons suffering from poliomyelitis (infantile paralysis), or of persons suspected of suffering from, or being subject thereto, and

Whereas, the use of such improperly prepared serum is imminently dangerous to public life, and

Whereas, such danger requires that stern preventive measures be immediately taken to protect the health and well-being of our people;

It is hereby ordered, that no public or private laboratory shall hereafter make or distribute convalescent or other serum of the character and for the purpose above noted, unless such laboratory be first approved as to equipment, training of personnel, technique, and process of manufacture by the State department of public health or its appointed agents.

This rule and regulation is hereby declared effective for 60 days, from and after this date.

STATE DEPARTMENT OF PUBLIC HEALTH,
By J. D. DUNSHEE, M.D., *Director*.

Dated this 27th day of June, 1934, at Sacramento, Calif.

COURT DECISION ON PUBLIC HEALTH

Injury resulting from injection of typhoid serum held compensable under workmen's compensation act.—(Louisiana Court of Appeal; *Smith* v. *Brown Paper Mill Co. Inc.*, 152 So. 700; decided Feb. 5, 1934.) An action was brought against an employer to recover compensation under the workmen's compensation act for injuries following an injection in the plaintiff's arm of typhoid serum, the inoculation having been given during working hours at the defendant company's first-aid room by a trained nurse employed by the company. A notice had been posted upon the company's bulletin board concerning employees reporting to the first-aid room for typhoid inoculations, and respecting this the court of appeals said:

> While it is clearly shown that its workmen were not ordered or required to take the shots at the plant, the wording of the posted notice may well have been interpreted by the average mill worker as such a requirement. In this case the plaintiff testifies that he so understood it and was so told by the company doctor. In any event the convenience of the facility and the posted notice constituted a suggestion, an invitation and urge, calculated to induce an employee to submit to the treatment who might not otherwise have done so. * * *
>
> If not compelled to take the treatment it was at least incidental to his employment.

The court also stated that, in its opinion, the disability "was caused by the destruction of the musculospiral nerve, not by serum sickness, but by the injection of the serum into or in the area of the nerve, causing direct injury or shock." "We are not prepared to say", said the court, "just what the nurse did, but it is evident that she accidentally and unintentionally gave this injection in a different manner from those given before to have caused the startlingly different result."

The appellate court took the view that the plaintiff was entitled to compensation under the workmen's compensation act, concluding that he had suffered an "accident", within the terms of the compensation act, arising out of and happening in the course of his employment.

DEATHS DURING WEEK ENDED JUNE 30, 1934

[From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce]

	Week ended June 30, 1934	Corresponding week, 1933
Data from 86 large cities of the United States:		
Total deaths	7,785	7,321
Deaths per 1,000 population, annual basis	10.8	10.2
Deaths under 1 year of age	564	526
Deaths under 1 year of age per 1,000 estimated live births	52	[1] 43
Deaths per 1,000 population, annual basis, first 26 weeks of year	12.1	11.5
Data from industrial insurance companies:		
Policies in force	67,791,606	67,779,572
Number of death claims	12,048	12,192
Death claims per 1,000 policies in force, annual rate	9.3	9.4
Death claims per 1,000 policies, first 26 weeks of year, annual rate	10.6	10.5

[1] Data for 81 cities.

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers

Reports for Weeks Ended July 7, 1934, and July 8, 1933

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 7, 1934, and July 8, 1933

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933
New England States:								
Maine	1		1		6	1	0	0
New Hampshire					65	6	0	0
Vermont	1				16	41	0	0
Massachusetts	10	11			299	413	0	1
Rhode Island		1			21	1	0	0
Connecticut	1	2	2	1	118	58	0	0
Middle Atlantic States:								
New York	28	36	[1] 1	[1] 3	429	607	2	1
New Jersey	18	20	1	3	442	323	3	1
Pennsylvania	30	27			856	420	4	2
East North Central States:								
Ohio	16	29	3		387	283	2	3
Indiana	3	7	1	13	67	34	0	1
Illinois	37	19	5	18	702	178	5	9
Michigan	7	21			179	171	0	0
Wisconsin	6	1	2	5	821	127	3	1
West North Central States:								
Minnesota	9	4	2		40	31	1	0
Iowa [2]		6			61	7	0	1
Missouri	19	23			51	64	0	1
North Dakota	3	2			59	29	1	0
South Dakota	2				36	4	0	0
Nebraska	4	4			7	45	0	0
Kansas	2	2			77	34	0	0
South Atlantic States:								
Delaware	1	2			13	6	0	0
Maryland [1 2 4]	2	11		1	149	8	0	0
District of Columbia	2	1		1	12	11	0	0
Virginia [4]	6	3			276	55	1	0
West Virginia	11	13	5	1	59	12	1	1
North Carolina [4]	6	12	1		142	74	0	0
South Carolina	1	5	53	115	36	66	0	0
Georgia [4]		8				50	0	0
Florida	6	4		1	67	19	0	0
East South Central States:								
Kentucky [2]	6	7	31		213	17	1	0
Tennessee	4	5	3	5	22	94	0	2
Alabama [4]	16	7		5	102	22	0	1
Mississippi [2]	6	2					0	0

Footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 7, 1934, and July 8, 1933—Continued

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933
West South Central States:								
Arkansas	1	5	3	3	5	68	1	1
Louisiana	13	10	3	6	50	9	0	0
Oklahoma [3]	3	10	3	6	25	11	1	0
Texas [4]	42	49	41	44	219	153	1	0
Mountain States:								
Montana [3]	1	1	6		10	17	0	0
Idaho [3]		1		1	3	1	0	0
Wyoming [3]	2				13	1	2	0
Colorado	3	3			398	5	0	0
New Mexico		5	8		13	6	1	0
Arizona	5	3	4		3	15	0	0
Utah [3]					4	27	0	0
Pacific States:								
Washington	1	1	1	1	69	32	3	1
Oregon	2	2	17	5	9	30	1	0
California	32	29	14	15	209	343	1	2
Total	369	414	211	253	6,860	4,119	35	29

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933
New England States:								
Maine	0	0	14	2	0	0	1	0
New Hampshire	1	0	2	14	0	0	0	0
Vermont	0	0		2	0	0	0	0
Massachusetts	0	7	71	93	0	0	1	3
Rhode Island	0	0	4	10	0	0	1	0
Connecticut	0	0	14	23	0	0	1	0
Middle Atlantic States:								
New York	7	4	173	177	0	0	5	31
New Jersey	2	0	41	48	0	0	16	6
Pennsylvania	2	0	177	177	0	0	14	8
East North Central States:								
Ohio	1	1	177	184	0	5	9	37
Indiana	0	1	31	21	0	1	5	5
Illinois	3	3	190	115	2	2	35	31
Michigan	0	0	113	158	0	1	5	3
Wisconsin	1	0	87	36	9	7	2	1
West North Central States:								
Minnesota	1	1	31	11	1	0	2	0
Iowa [3]	0	1	17	11	4	6	3	0
Missouri	0	1	25	13	2	1	23	5
North Dakota	0	1	4	2	1	2	1	1
South Dakota	1	0	1	5	0	0	0	0
Nebraska	0	0	8	14	4	6	0	1
Kansas	1	0	15	8	0	0	10	16
South Atlantic States:								
Delaware	0	0	2	3	0	0	0	3
Maryland [2 3 4]	1	3	15	22	0	0	9	11
District of Columbia	0	0	7	3	0	0	1	0
Virginia [3]	1	0	15	17	0	0	18	49
West Virginia	3	0	18	13	0	0	11	20
North Carolina [3]	0	0	11	14	0	0	6	37
South Carolina	0	0	1		0	0	19	47
Georgia [4]	0	0	1	5	0	0	39	46
Florida	0	0			0	0	2	0
East Central States:								
Kentucky [3]	5	1	22	9	3	0	79	65
Tennessee	1	5	8	15	0	0	21	75
Alabama [4]	0	0	5	12	0	0	20	34
Mississippi [3]	0	1	7	8	0	1	16	23

Footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended July 7, 1934, and July 8, 1933—Continued

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933	Week ended July 7, 1934	Week ended July 8, 1933
West South Central States:								
Arkansas	0	2	1	2	0	0	21	26
Louisiana	0	1	2	4	0	0	7	27
Oklahoma [5]	0	0	3	7	1	0	19	25
Texas [4]	5	3	30	33	14	9	66	64
Mountain States:								
Montana [2]	3	0	5	7	0	0	2	5
Idaho [2]	4	0	1		0	0	0	0
Wyoming [2]	0	0	17	6	7	1	0	0
Colorado	0	0	10	5	0	1	5	2
New Mexico	0	0	2	2	0	0	5	1
Arizona	2	0		3	0	1	4	1
Utah [2]	1	0	2	3	0	0	0	3
Pacific States:								
Washington	2	0	18	20	11	6	3	1
Oregon	2	1	19	10	1	2	2	8
California	266	3	79	88	1	8	9	6
Total	316	40	1,496	1,435	61	60	518	727

[1] New York City only.
[2] Week ended earlier than Saturday.
[3] Rocky Mountain spotted fever, week ended July 7, 1934, 14 cases, as follows: Maryland, 2; Virginia, 4; North Carolina, 2; Kentucky, 1; Montana, 2; Idaho, 1; Wyoming, 2.
[4] Typhus fever, week ended July 7, 1934, 40 cases, as follows: Maryland, 1; Georgia, 7; Alabama, 8; Texas, 24.
[5] Exclusive of Oklahoma City and Tulsa.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week:

State	Meningococcus meningitis	Diphtheria	Influenza	Malaria	Measles	Pellagra	Poliomyelitis	Scarlet fever	Smallpox	Typhoid fever
May 1934										
Mississippi	1	22	834	5,869	3,990	527	1	19	2	109
Nevada			4		300		0	12	0	3
North Carolina	6	55	136		6,907	68	2	71	3	7
Tennessee	10	24	131	148	1,565	21	0	77	2	16
June 1934										
Connecticut	4	16	2	1	837			120	0	1
Indiana	3	40	39		2,083			233	6	36
Iowa	4	24		1	679		1	130	3	5
Nebraska		23			319		1	64	16	7

May 1934

	Cases
Chicken pox:	
Mississippi	485
Nevada	37
North Carolina	344
Tennessee	85
Dysentery:	
Mississippi (amoebic)	82
Tennessee	13
German measles:	
North Carolina	140
Tennessee	38
Hookworm disease:	
Mississippi	325
Impetigo contagiosa:	
Tennessee	4

May 1934—Contd.

	Cases
Lethargic encephalitis:	
Tennessee	4
Mumps:	
Mississippi	464
Nevada	2
Tennessee	100
Ophthalmia neonatorum:	
Tennessee	5
Paratyphoid fever:	
North Carolina	1
Tennessee	1
Puerperal septicemia:	
Mississippi	22
Rabies in animals:	
Mississippi	6

May 1934—Contd.

	Cases
Rocky Mountain spotted fever:	
Nevada	1
North Carolina	1
Septic sore throat:	
North Carolina	5
Tennessee	15
Tetanus:	
Tennessee	2
Trachoma:	
Mississippi	11
Tennessee	11
Tularaemia:	
Nevada	1
North Carolina	1
Tennessee	1

May 1934—Contd.

	Cases
Undulant fever:	
Mississippi	2
North Carolina	1
Vincent's infection:	
Tennessee	3
Whooping cough:	
Mississippi	1,057
Nevada	21
North Carolina	1,699
Tennessee	190

June 1934

Chicken pox:	
Connecticut	505
Indiana	80
Iowa	123
Nebraska	87
Conjunctivitis:	
Connecticut	1

June 1934—Contd.

	Cases
Dysentery:	
Connecticut (bacillary)	2
German measles:	
Connecticut	33
Iowa	109
Lead poisoning:	
Connecticut	1
Mumps:	
Connecticut	207
Indiana	13
Iowa	94
Nebraska	30
Paratyphoid fever:	
Connecticut	1
Rabies in animals:	
Connecticut	1
Indiana	33
Rabies in man:	
Iowa	1

June 1934—Contd.

	Cases
Rocky Mountain spotted fever:	
Iowa	1
Septic sore throat:	
Connecticut	12
Iowa	9
Nebraska	14
Trachoma:	
Iowa	1
Trichinosis:	
Connecticut	2
Undulant fever:	
Connecticut	7
Indiana	3
Iowa	7
Nebraska	2
Whooping cough:	
Connecticut	268
Indiana	265
Iowa	141
Nebraska	96

PLAGUE-INFECTED RODENTS IN MODOC COUNTY, CALIF.

The Director of Public Health of California has reported plague-infected ground squirrels in Modoc County, Calif., as follows: June 26, 1934, 6; July 5, 1934, 1; July 9, 1934, 1.

WEEKLY REPORTS FROM CITIES

City reports for week ended June 30, 1934

This table summarizes the reports received regularly from a selected list of 121 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Maine:											
Portland	0	----	1	0	4	8	0	0	1	12	20
New Hampshire:											
Concord	0	----	0	1	2	1	0	0	0	0	11
Nashua	0	----	0	8	----	0	0	----	0	0	----
Vermont:											
Barre	0	----	0	0	0	0	0	2	0	0	6
Burlington	1	----	0	5	0	0	0	0	0	0	5
Massachusetts:											
Boston	5	----	1	149	11	31	0	11	0	42	179
Fall River	0	----	0	0	0	1	0	0	0	1	22
Springfield	0	----	0	0	0	1	0	0	0	7	28
Worcester	2	----	0	2	0	11	0	2	0	17	41
Rhode Island:											
Pawtucket	0	----	0	0	0	0	0	0	0	0	11
Providence	0	----	0	18	2	4	0	5	1	25	60
Connecticut:											
Bridgeport	0	----	0	0	0	4	0	0	0	3	19
Hartford	1	----	0	19	1	1	0	2	0	2	39
New Haven	0	----	0	1	2	1	0	1	0	4	47
New York:											
Buffalo	0	----	0	44	4	15	0	4	0	11	105
New York	14	3	3	172	82	84	0	86	9	146	1,300
Rochester	0	----	0	3	3	21	0	1	0	5	51
Syracuse	0	----	0	40	2	2	0	0	0	52	38
New Jersey:											
Camden	1	----	0	1	0	4	0	1	0	1	18
Newark	0	3	0	15	3	3	0	9	0	34	88
Trenton	0	----	0	2	1	12	0	2	0	0	37
Pennsylvania:											
Philadelphia	4	----	0	43	10	35	0	29	0	122	429
Pittsburgh	6	2	1	132	6	23	0	12	1	23	154
Reading	0	----	0	0	1	0	0	0	0	9	28
Scranton	0	----	----	11	----	3	0	----	1	13	----

City reports for week ended June 30, 1934—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Ohio:											
Cincinnati	1	----	1	3	6	4	0	3	0	7	130
Cleveland	2	8	0	274	10	24	0	16	0	68	186
Columbus	1	----	0	0	3	17	0	5	0	19	82
Toledo	1	1	0	47	2	20	0	5	0	62	63
Indiana:											
Fort Wayne	2	----	0	1	0	2	0	1	2	0	22
Indianapolis	0	----	0	36	6	5	0	4	0	14	----
South Bend	0	----	0	6	1	2	0	0	0	0	16
Terre Haute	0	----	0	0	0	0	0	1	1	0	25
Illinois:											
Chicago	14	----	1	442	30	123	0	47	22	108	662
Springfield	1	----	0	6	0	0	0	0	0	14	19
Michigan:											
Detroit	3	----	0	61	4	32	0	20	0	154	223
Flint	0	----	0	0	3	10	0	1	0	12	22
Grand Rapids	0	----	0	4	0	7	0	1	0	5	27
Wisconsin:											
Kenosha	0	----	0	11	0	0	0	0	0	2	10
Madison	0	----	0	35	0	0	0	0	0	8	13
Milwaukee	3	2	2	232	0	102	0	4	0	70	95
Racine	1	----	0	2	1	8	0	1	0	7	15
Superior	0	----	0	4	1	0	0	0	0	0	5
Minnesota:											
Duluth	0	----	----	0	----	0	0	----	0	1	27
Minneapolis	3	----	0	1	1	12	0	2	0	4	90
St. Paul	1	----	0	6	4	4	0	1	0	15	57
Iowa:											
Davenport	0	----	----	4	----	0	0	----	0	0	----
Des Moines	0	----	----	8	----	9	0	----	0	0	40
Sioux City	0	----	----	8	----	0	0	----	0	5	----
Waterloo	0	----	----	2	----	0	1	----	0	3	----
Missouri:											
Kansas City	2	----	0	2	11	6	0	9	0	17	113
St. Joseph	3	----	0	0	1	3	0	1	1	1	35
St. Louis	11	----	0	0	10	4	0	19	4	66	281
North Dakota:											
Fargo	0	----	0	0	0	0	0	0	0	14	6
South Dakota:											
Aberdeen	0	----	----	14	----	0	0	----	0	13	----
Nebraska:											
Omaha	1	----	0	3	2	8	1	3	0	5	66
Kansas:											
Topeka	0	----	0	18	1	4	0	0	1	40	15
Wichita	3	----	0	6	1	0	0	1	0	2	24
Delaware:											
Wilmington	0	----	0	2	----	0	0	----	0	1	----
Maryland:											
Baltimore	1	1	1	114	14	16	0	13	3	104	208
Cumberland	0	----	0	2	0	0	0	2	0	0	10
Frederick	0	----	0	0	0	0	0	0	0	0	3
District of Columbia:											
Washington	1	1	1	18	8	5	0	10	0	0	137
Virginia:											
Lynchburg	0	----	0	75	0	1	0	0	0	26	16
Norfolk	0	----	0	0	0	2	0	4	0	48	53
Richmond	1	----	0	39	1	2	0	4	1	0	63
Roanoke	0	----	0	2	0	0	0	1	4	0	13
West Virginia:											
Charleston	1	----	0	15	0	0	0	1	0	1	11
Huntington	0	----	----	1	----	1	0	----	0	1	----
Wheeling	0	----	0	2	1	4	0	1	2	2	21
North Carolina:											
Raleigh	0	----	0	0	1	0	0	0	0	8	20
Wilmington	0	----	0	1	0	0	0	0	0	16	19
Winston-Salem	1	----	0	0	0	4	0	5	1	15	23
South Carolina:											
Charleston	0	6	0	1	2	0	0	2	0	0	24
Columbia	0	----	0	0	5	0	0	0	0	0	31
Georgia:											
Atlanta	1	----	0	1	1	0	0	5	2	10	99
Brunswick	0	----	0	2	0	0	0	0	0	0	----
Savannah	0	4	0	0	2	0	0	6	0	5	26
Florida:											
Miami	0	----	0	14	1	0	0	1	0	3	23
Tampa	1	----	0	9	1	0	0	2	0	0	19
Kentucky:											
Ashland	0	----	----	10	----	1	0	----	0	6	----
Lexington	0	----	0	15	1	0	0	3	0	10	18
Louisville	1	----	0	131	3	3	0	1	1	18	75

City reports for week ended June 30, 1934—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Smallpox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
Tennessee:											
Memphis	0	----	0	7	4	1	0	5	1	18	100
Nashville	2	----	0	1	4	4	0	0	0	4	55
Alabama:											
Birmingham	1	----	1	25	6	1	0	5	2	10	35
Mobile	1	----	0	1	0	1	0	2	2	5	20
Montgomery	0	----	----	0	----	1	0	----	0	1	----
Arkansas:											
Fort Smith	0	----	----	0	----	0	0	----	0	0	----
Little Rock	0	----	0	0	4	0	0	2	1	0	----
Louisiana:											
New Orleans	10	2	2	5	4	8	0	15	8	2	106
Shreveport	0	----	0	1	3	0	0	0	0	5	50
Oklahoma:											
Oklahoma City	0	3	0	0	5	0	0	1	0	2	69
Texas:											
Dallas	4	----	0	0	1	2	0	4	2	23	67
Fort Worth	0	----	0	2	1	1	0	0	1	2	36
Galveston	0	----	0	0	0	0	1	0	0	0	10
Houston	2	----	0	1	1	3	0	6	3	1	70
San Antonio	0	----	0	0	7	0	0	7	0	0	72
Montana:											
Billings	1	----	0	0	0	0	0	0	0	7	7
Great Falls	0	----	0	----	1	0	0	0	1	1	11
Helena	0	----	0	0	0	0	0	0	0	0	6
Missoula	0	----	0	0	0	0	0	0	0	0	12
Idaho:											
Boise	0	----	0	1	0	1	0	0	0	1	4
Colorado:											
Denver	6	----	0	245	4	10	0	1	1	48	73
Pueblo	0	----	0	45	2	0	0	0	0	4	12
New Mexico:											
Albuquerque	0	----	0	1	0	2	0	2	0	3	11
Utah:											
Salt Lake City	0	----	0	2	2	6	0	0	1	121	35
Nevada:											
Reno	0	----	0	0	0	0	0	0	0	0	5
Washington:											
Seattle	0	----	0	27	3	6	7	5	0	27	70
Spokane	0	----	0	5	1	1	0	0	0	14	26
Tacoma	0	----	0	15	2	2	0	0	0	7	31
Oregon:											
Portland	0	----	0	6	1	7	0	1	1	11	56
Salem	0	----	----	0	----	0	0	----	0	3	----
California:											
Los Angeles	16	10	0	19	12	29	0	19	1	31	----
Sacramento	0	----	0	5	4	0	0	3	2	2	33
San Francisco	0	1	1	188	4	2	0	6	1	8	140

State and city	Meningococcus meningitis		Poliomyelitis cases
	Cases	Deaths	
Massachusetts:			
Boston	1	0	0
Springfield	1	0	0
New York:			
New York	3	0	0
New Jersey:			
Camden	1	0	1
Pennsylvania:			
Pittsburgh	1	0	1
Ohio:			
Cincinnati	0	2	0
Toledo	1	1	0
Illinois:			
Chicago	6	3	4
Michigan:			
Grand Rapids	1	0	0
Wisconsin:			
Madison	1	0	0
Milwaukee	0	0	1
Missouri:			
St. Joseph	1	0	0
St. Louis	2	2	0
District of Columbia:			
Washington	0	1	0
Florida:			
Miami	0	0	1
Kentucky:			
Louisville	0	1	0
Louisiana:			
New Orleans	1	0	0
Idaho:			
Boise	0	0	1
Colorado:			
Denver	0	1	0
Washington:			
Spokane	2	1	0
Oregon:			
Portland	0	0	3
California:			
Los Angeles	0	0	126
Sacramento	0	0	1
San Francisco	0	0	15

Lethargic encephalitis.—Cases: Newark, N.J., 1; Pittsburgh, 2; Chicago, 1; Detroit, 1; Kansas City, Mo., 1.
Pellagra.—Cases: Philadelphia, 1; Baltimore, 1; Raleigh, 1; Charleston, S.C., 1; Atlanta, 1; Brunswick, 1; Savannah, 1; Mobile, 1; Little Rock, 1; New Orleans, 1; Dallas, 1.
Typhus fever.—Cases: Tampa, 1; Birmingham, 1.

FOREIGN AND INSULAR

CANADA

Quebec Province—Communicable diseases—2 weeks ended June 30, 1934.—The Bureau of Health of the Province of Quebec, Canada, reports cases of certain communicable diseases for the 2 weeks ended June 30, 1934, as follows:

Disease	Cases	Disease	Cases
Cerebrospinal meningitis	1	Measles	568
Chicken pox	137	Poliomyelitis	1
Diphtheria	24	Puerperal septicemia	1
Dysentery	2	Scarlet fever	113
Erysipelas	6	Tuberculosis	147
German measles	5	Typhoid fever	16
Influenza	1	Undulant fever	1
Lethargic encephalitis	1	Whooping cough	149

CZECHOSLOVAKIA

Communicable diseases—April 1934.—During the month of April 1934 certain communicable diseases were reported in Czechoslovakia, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Anthrax	4	----	Paratyphoid fever	8	----
Cerebrospinal meningitis	13	6	Poliomyelitis	5	1
Chicken pox	305	----	Puerperal fever	46	14
Diphtheria	1,805	114	Scarlet fever	1,719	18
Dysentery	6	1	Trachoma	136	----
Influenza	69	12	Typhoid fever	302	30
Malaria	124	----	Typhus fever	78	2

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

(NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for June 29, 1934, pp. 768-781. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued July 27, 1934, and thereafter, at least for the time being, in the issue published on the last Friday of each month.)

Cholera

Philippine Islands—Manila.—During the week ended June 30, 1934, 1 case of cholera was reported in Manila, Philippine Islands.

Plague

Egypt—Minufiya Province.—During the week ended June 30, 1934, 1 case of plague was reported in Minufiya Province, Egypt.

Indo-China.—For the week ended June 30, 1934, plague has been reported in Indo-China, as follows: 1 case at Longxuyen, 1 case at Sadec, and 1 case at Vinhlong.

Libya.—For the period June 11–20, 1934, 5 cases of plague with 1 death were reported in Libya.

United States—California.—A report of plague in California appears on page 863 of this issue of PUBLIC HEALTH REPORTS.

Smallpox

Egypt—Damietta Province.—During the week ended June 23, 1934, 1 case of smallpox with 1 death was reported in Damietta Province, Egypt.

Typhus Fever

Spain—Catalonia.—During the week ended June 16, 1934, 27 cases of typhus fever were reported in Catalonia, Spain.

Yellow Fever

Ivory Coast—Abidjan.—On June 26, 1934, 1 case of yellow fever with 1 death was reported in Abidjan, Ivory Coast.